

4.0 BIOPHYSICAL ISSUES

4.1 BIOPHYSICAL LITERATURE

- 1 See the NIEHS review and Appendix B. The NIEHS Working Group (1999) has reviewed relevant biophysical discussions where pro and con arguments are summarized below.
- 2 (IMPORTANT NOTE: Table 4.1.1. and all the following similar tables are meant to be as comprehensive as possible. The reviewers have strived to include ALL
- 3 conceivable arguments that can be raised in favor or against the hypothesis of causality, whether based on data or on speculation. Inclusion of an argument does not
- 4 necessarily mean that that argument is supported by any of the reviewers. The reviewers' judgment is expressed only in the third column, "COMMENT AND
- 5 SUMMARY.")

TABLE 4.1.1 BIOPHYSICAL PRO AND CON ARGUMENTS

BIOPHYSICAL PRO AND CON ARGUMENTS		
AGAINST CAUSALITY	FOR CAUSALITY	COMMENT AND SUMMARY
General		
(A1) All biological models of hypothesized mechanisms (e.g., magnetite) show that no effects are possible at environmental levels.	(F1) One cannot anticipate all the possible biological structures and configurations occurring within the body at the molecular, cellular, and organ levels. The physics of these models may be correct, but the biological assumptions are simple and perhaps incomplete. Thus it is impossible to predict what is and is not possible.	(C1) A credible biophysical-mechanism hypothesis would boost the level of confidence tremendously, but absence of one cannot be used to dismiss empirical epidemiological evidence.
(A2) Forces and energies involved in biochemical processes are far stronger than those induced in humans by environmental fields.	(F2) Power frequency fields exhibit spatial and temporal coherence that may make them discernable above the random endogenous noise.	(C2) This argument has already been considered in setting the prior; therefore, it cannot be used to modify it.

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<p>(A3) The resonance mechanisms are not supported by common sense argument. They assume molecules or atoms without surrounding molecules. No resonance model has been replicated reliably in multiple laboratories.</p> <p>(A3a) The theories led to epidemiological validation (Bowman et al., 1995), (Kaune, 1994b), (Kaune et al., 2002) with conflicting results.</p>	<p>(F3) Several models have been proposed that may well be viable considering the fact that biological processes depend on continuous energy input and therefore cannot be adequately described by models based on equilibrium thermodynamics.</p> <p>Several of these models (e.g., cyclotron resonance and parametric resonance) are supported by some in vitro data.</p> <p>(F3a) Some analyses suggest a weak agreement between Kaune and Bowman. Better personal exposure monitoring may show an effect.</p>	<p>(C3) Having a clear or even simplified, but uncontroverted, mechanism would strongly increase the posterior. However, given the complexity of the characteristics of the exposure, the nature of biological processes, and the ill-understood etiologies of the diseases associated with EMF exposure, the fact that these mechanisms are still tentative and controversial cannot be used as an argument against causality.</p> <p>(C3a) While it is possible that brief flashes of resonance could occur when the right combination of alternating (AC) and steady (DC) fields are encountered, given the demonstrated variability of both fields in the residential environment, it is hard to believe that the associations seen to date, which based on measurements taken in one location, could be strongly correlated with personal exposures. In any case, resonance conditions are not associated with wire code or high TWA magnetic fields and thus do not explain their associations with disease.</p>
<p>(A4) The field itself grows, collapses, and then grows in the opposite direction and collapses 50-60 times a second. So, the average field is always zero. Therefore, for basic symmetry principles, effects of 50-60 Hz EMF should vary as the square of the intensity. The reviewers have an upper benchmark for biological effects from which they can infer the shape of the lower end of the theoretically proper dose response, which is based on the square of the field, [the phenomenon of phosphenes (flashes of light) induced by magnetic fields at the Tesla level]. The human epidemiology does not follow the predicted shape and thus must be due to bias or confounding.</p>	<p>(F4) Many materials (including cell membranes) exhibit nonlinear electrical properties; therefore symmetry arguments do not apply. In interaction where the time scale is short relative to the period of the applied signal, the above arguments for a B-squared dependence are not relevant. For example, a neuron that fires rhythmically at 100 Hz would experience only part of a 60 Hz cycle before firing. The average value of this part of cycle is not zero.</p> <p>Even if the initial interaction depends on the square of the field, there is no reason to believe that in the complex chain of events between this first step and the manifestation of a disease, this square field relationship should be retained.</p> <p>A physical agent may interact in more ways than one. The phosphene phenomenon may not be the proper anchor for a carcinogenic or reproductive</p>	<p>(C4) Prediction and evaluation of evidence is fine when one understands the system being evaluated, which is usually the case in physics.</p> <p>There is too much scientists do not understand to give weight to predictions about dose response based on simple physical principles.</p>

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	health process.	
(A5) Attempts to use theory to predict effects have not been productive.	(F5) Most of the biophysical theorizing has not reflected close collaboration with experimentalists.	(C5) Until there are accepted robust effects at levels below 100 mG, where current theories suggest no effects are possible, there can be no evidence on which to try out theories.
(A6) The strategy of physics, to predict results from first principles and then test them, is time tested and successful. It predicts that EMF effects are impossible at residential levels of exposure.	<p>(F6a) To use theory to predict empirical observation is only ONE of the strategies of physics and not the mainstay of modern science, in which observation is the ultimate test of truth.</p> <p>(F6b) Over the two decades of EMF research, the calculated threshold for EMF interaction has decreased as the biological component of the models has become more sophisticated. This argues that these thresholds cannot yet be accepted as accurate.</p>	(C6) Theory can guide experimentation when the system is sufficiently understood. The changing predictions remind the reviewers how little this system is understood.
(A7) There are no published robust experimental effects seen in multiple laboratories, at levels below 40-100 mG, which is what theory predicted.		(C7) The chicken embryo literature shows statistically significant effects in the 40–100 mG range, which have been dismissed because the effect was not larger than the variation between historical controls. This is an additional evidentiary condition imposed by regulatory agencies to avoid false positives. The reviewers do not totally ignore this evidence.
		(C8) The demand that experimental mechanistic effects be detectable at residential levels of exposure is a stringent requirement that many recognized chemical pathogens would not be able to meet.

4.2 CONCLUSIONS

1 While biophysical arguments seem to have strongly decreased the confidence of
2 potential health effects of some scientists (primarily physicists), these arguments did
3 not influence to any great degree the initial degree of confidence or the updated
4 degree of confidence of the review team. The fact that chicken embryo experiments
5 appear to offer some evidence contrary to the theoretical predictions increases our

6 skepticism in theoretical models. Overall, the prior of the review team was little
7 changed by biophysical arguments.